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Major genes for resistance to beet necrotic yellow vein virus (BNYVV) in *Beta vulgaris* *

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Summary

Inheritance of resistance to beet necrotic yellow vein virus (BNYVV) was studied in segregating F2 and backcross families obtained from crosses between resistant plants of the sugar beet selection Holly-1-4 or the wild beet accession *Beta vulgaris* subsp. *maritima* WB42 and susceptible parents. Greenhouse tests were carried out, in which seedlings were grown in a mixture of sand and infested soil. Virus concentrations of BNYVV in the rootlets were estimated by ELISA. To discriminate resistant and susceptible plants, mixtures of normal distributions were fitted to $\log_{10}$ virus concentrations, estimated for segregating F1, F2 and BC populations of both accessions. The hypothesis that Holly-1-4 contained one single dominant major gene was accepted. For WB42, results fitted with the hypotheses that resistance was based on either one (or more) dominant major gene(s) showing distorted segregation, or two complementary dominant genes, which are both required for resistance. Resistance from WB42 appeared to be more effective against BNYVV than resistance from Holly-1-4.

Introduction

Infections with beet necrotic yellow vein virus (BNYVV), the causal agent of rhizomania in sugar beet (*Beta vulgaris* L.) (Tamada, 1975), can lead to severe losses in root yield and sugar content (Johansson, 1985; Richard-Molard, 1985). The thick-walled resting spores of the soil-borne fungus *Polymyxa betae* Keskin, the vector of BNYVV can remain infective in the soil and transmit the virus for at least fifteen years (Abe & Tamada, 1986), making crop rotation ineffective to control the disease. Therefore, the cultivation of cultivars with resistance to rhizomania on infested soils is the most promising way to control rhizomania in sugar beets (Schlösser, 1988; Asher, 1993). Resistance to BNYVV was described for accessions of *B. vulgaris* subsp. *vulgaris, B. vulgaris* subsp. *maritima* (L.) Arcang. (Lewellen et al., 1987; Whitney, 1989), *B. corolliflora* Zos., *B. intermedia* Bunge and *B. lomatogona* Fisch. & Mey. (Paul et al., 1993b). Some of these accessions also contained resistance to *P. betae* (Fujisawa & Sugimoto, 1979; Paul et al., 1993b). The perspective to use resistance to *P. betae* seems to be limited, however, because plants were found which contained no or only low numbers of resting spores, but high virus concentrations (Paul et al., 1993b).

The *B. vulgaris* sugar beet accession Holly and the wild beet accession *B. vulgaris* subsp. *maritima* WB42 were found to be resistant to rhizomania (Lewellen et al., 1987; Whitney, 1989). Lewellen et al. (1987) suggested that the resistance in Holly is simply inherited and possibly conditioned by a single dominant gene, but the observed segregation data did not always fit the expected ratios. Field tests were also performed with progenies of crosses between sugar beet and WB42, which appeared to be either uniformly resistant or segregated for resistance and susceptibility. Resistance of the subsp. *maritima* was shown to be dominant, but it

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Table 1. Estimated mixture model parameters based on $\log_{10}$ BNYVV concentrations (in ng/ml) in rootlets of individual plants of the resistant accessions Holly-1-4 and WB42, the susceptible accessions 'Queen', MS-2 and crosses between resistant and susceptible plants

Plant materials[a]	n	Mean low virus concentration of resistant plants	Mean high virus concentration of susceptible plants	SD
Parents				
Holly-1-4	48	1.50		0.21
WB42	88	0.68	2.19	0.28
'Queen'	48		2.82	0.18
MS-2	48		2.65	0.19
Crosses with Holly-1-4[b]:				
F1(91.11)	48	1.56		0.21
F1(91.38)	32	1.63		0.25
F2(92.01)	80	1.29	2.51	0.30
F2(92.03)	80	1.54	2.45	0.29
F2(92.11)	79	1.34	2.52	0.26
F2(92.13)	32	1.37	2.09	0.30
BC(92.03)	135	1.35	2.47	0.28
BC(92.13)	31	1.26	2.18	0.21
Crosses with WB42[c]:				
F1(91.05)	57	0.93		0.28
F1(91.14)	64	1.26	2.53	0.35
F1(91.32)	47	0.71	2.46	0.38
F2(92.04)	79	0.90	2.06	0.40
F2(92.06)	32	0.82	2.10	0.41
F2(92.14)	48	0.94	2.07	0.30
F2(92.26)	31	0.64	1.77	0.32
F2(93.38)	56	0.73	2.09	0.32
F2(93.39)	32	0.70	2.17	0.39
BC(92.06)	32	1.50	2.17	0.18
BC(92.26)	224	1.15	2.18	0.29
BC(93.38)	32	0.87	2.06	0.26
BC(93.39)	32	0.85	2.04	0.30
Control				
'Regina'	88		2.57	0.19

[a] Identification number of the crosses in parentheses.
[b] Resistant F1 plants were selected from F1(91.11) for the production of F2 and BC families.
[c] Resistant F1 plants were selected from F1(91.14) for the production of F2 and BC families 92.04, 92.06, 92.14 and 92.26, and from F1(91.05) for 93.38 and 93.39.

remained unclear if it was conditioned by one or a few major genes (Lewellen et al., 1987; Whitney, 1989).

In greenhouse tests, the virus replicates at a considerable lower rate in both Holly-1-4, a selection of the Holly source (Lewellen et al., 1987) and WB42 (Paul et al., 1993b). Other studies (Paul et al., 1993c; Scholten et al., 1994) demonstrated that the mechanisms of resistance to BNYVV in Holly differed from that in WB42. The combination of different mechanisms of resistance in commercial hybrids may be advantageous to provide higher levels of resistance and to improve the durability of a single dominant major gene for resistance (Lewellen & Biancardi, 1990). The aim of the present study was to elucidate the inheritance of resistance to BNYVV in the selections of Holly-1-4 and especially of WB42 through analysis of segregating F2 and BC populations of both accessions. The effect of the resistances from both accessions was also compared.

Materials and methods

Plant materials and crosses

Studies on the inheritance of resistance to BNYVV were performed with the resistant sugar beet accession *B. vulgaris* Holly-1-4, which is a selection from the Holly source (Lewellen et al., 1987) and the resistant wild beet accession *B. vulgaris* subsp. *maritima* WB42. The Holly-1-4 selection originated from a bulk multiplication of plants obtained by selfing of one resistant inbred Holly plant. The WB42 selection consisted of a bulk multiplication of several WB42 plants. Both accessions are diploid with 2n = 18. To obtain F1 families, plants of both selections were crossed in pairs with plants of the susceptible red table beet *B. vulgaris* 'Queen' as a pollinator, resulting in red F1 plants, as the responsible major genes for colour inherit dominantly (Knapp, 1958; Wolyn & Gabelman, 1989). Segregating backcross (BC) families were obtained after crossing resistant F1 plants with plants of the susceptible male-sterile *B. vulgaris* MS-2. Selfing of F1 plants led to the production of F2 seed.

Greenhouse tests

To discriminate between resistant and susceptible plants, healthy seedlings were transplanted into a mixture of sand and infested soil and grown for one month as described previously (Paul et al., 1992). Purified virus, serially diluted in a solution of healthy plant sap and PBS-Tween 20 (1:20 v/v), was used in ELISA to estimate the virus concentrations in ng/ml in rootlets of individual plants (Paul et al., 1992). The $\log_{10}$ of the virus concentrations were used for statistical analysis. The detection limit for virus was at a $\log_{10}$ virus concentration of 0.65. The cultivar 'Regina' was used as a susceptible control in all greenhouse tests.

Mixture models

Many plants could be assigned to classes containing either low or high virus concentrations. However, plants with intermediate levels of resistance were also found. For the resistant accessions Holly-1-4, the susceptible accessions MS-2, 'Queen' and 'Regina' it was assumed that the $\log_{10}$ virus concentrations follow a normal distribution. Therefore, mixtures of normal distributions (Jansen, 1993, 1994) were then fitted to the F1, F2 and BC populations. Each component in the mixture corresponds to an underlying genotype. To assess major gene activity, the likelihood of the normal (non mixture) model was compared with the likelihoods of normal mixture models with two or more underlying components.

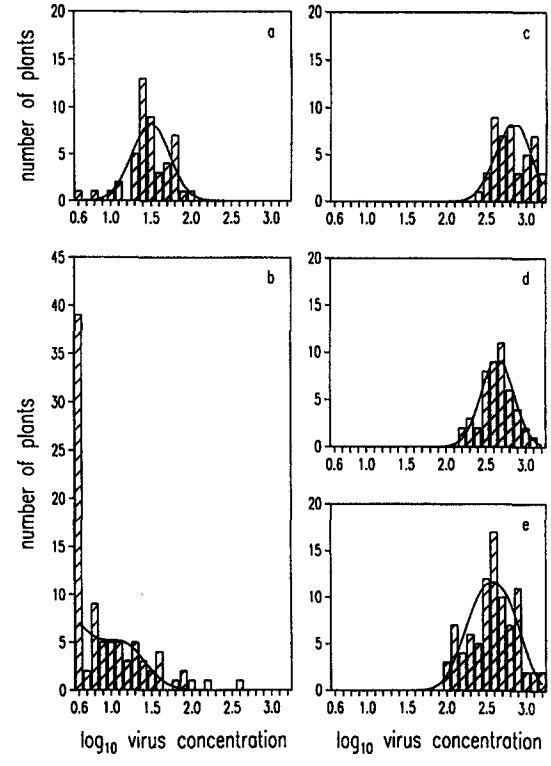

Figure 1. Curves fitted to histograms of $\log_{10}$ BNYVV concentrations of plants of the parental families Holly-1-4 (a), WB42 (b), 'Queen' (c), MS-2 (d), and the susceptible control cultivar 'Regina' (e).

Results

Virus concentrations in the resistant and susceptible parents

The average virus concentrations were estimated by ELISA in the rootlets of resistant and susceptible parents (Table 1). The Holly-1-4 selection was resistant with an average concentration of $\log_{10}$ 1.38, versus $\log_{10}$ 0.68 for the resistant accession WB42. About 44% of the WB42 plants were free of virus or contained levels below the detection limit, whereas about 7% of the plants contained $\log_{10}$ virus concentrations over 2.00, which were as high as those observed for the susceptible parents 'Queen', MS-2 and the susceptible

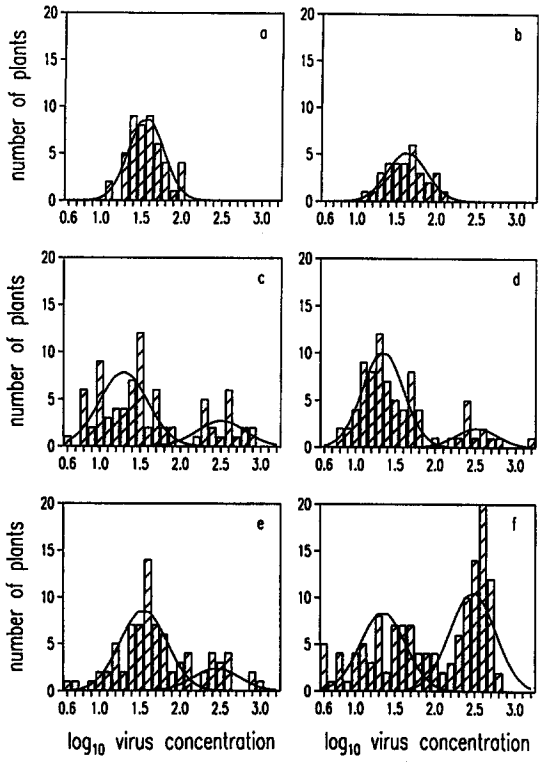

Figure 2. Curves fitted to histograms of $\log_{10}$ BNYVV concentrations of individual plants originating from crosses with Holly-1-4: F1 (91.11) (a), F1 (91.38) (b), F2 (92.01) (c), F2 (92.11) (d), F2 (92.03) (e) and BC (92.03) (f).

control 'Regina'. These plants were therefore classified as susceptible. Curves fitted to the data are shown in Figure 1 and demonstrate that the curve fitting for WB42 is distorted due to the high number of plants with no or hardly any virus, while the other parental accessions and also 'Regina' showed a normal distribution.

Virus concentrations in progenies from crosses with Holly-1-4

The two F1 progenies of Holly-1-4 studied consisted only of plants with low virus concentrations and were consequently classified as resistant (Table 1, Figure 2). The virus concentrations were in the same order of magnitude as those of the resistant parent Holly-1-4. The F2 and BC families segregated into plants with low virus concentrations, which did not differ significantly from Holly-1-4 and the F1 progenies, and high virus concentrations, as found in the susceptible parents (Table 1). The crossing point at which the curves of resistant and susceptible plants of the segregating families of Holly-1-4 cut each other was estimated around a $\log_{10}$ virus concentration of 2.0 (Figure 2).

Virus concentrations in progenies from crosses with WB42

WB42 consisted mostly of highly resistant plants. However, also a few susceptible plants were found. The selection of WB42 originated from a multiplication of several plants in bulk. Therefore, various genotypes of resistance to BNYVV could be expected, producing both resistant and segregating F1 progenies. Results of three F1 progenies tested confirmed this expectation as only F1 (91.05) was resistant, while the other two F1 families segregated into classes of plants with low and high virus concentrations (Table 1). Curves fitted to the data obtained from crosses with WB42 are shown in Figure 3. The F1 families contained 13–17% of plants in which no virus could be detected. F2 families segregated into groups of plants with low and high virus concentrations, which did not differ significantly from those in the segregating F1 families, and consisted of 8–28% plants without virus. Higher numbers of susceptible plants than resistant plants were found in the BC families. For the segregating progenies, including WB42, the crossing point at which the curves of resistant and susceptible plants cut each other was estimated around a $\log_{10}$ virus concentration of 1.5 ng/ml, which was somewhat lower than for segregating families of Holly-1-4.

Genetical analysis of resistance in Holly-1-4

Resistance from Holly-1-4 inherits dominantly, since both F1 progenies were resistant. They had similar virus concentrations in the rootlets as the resistant parent. Segregation ratios of resistant and susceptible plants based on mixture models together with the 95% confidence intervals are presented in Table 2. The hypothesis that resistance is based on one (or more) dominant major gene(s) was accepted for all F1, F2 and BC families, except for F2 (92.11) which contained just a few more resistant plants than expected.

Genetical analysis of resistance in WB42

To study the inheritance of resistance to BNYVV in WB42, the following possible genetical hypotheses were analyzed: the resistant phenotype is controlled by either a single dominant major gene for resistance,

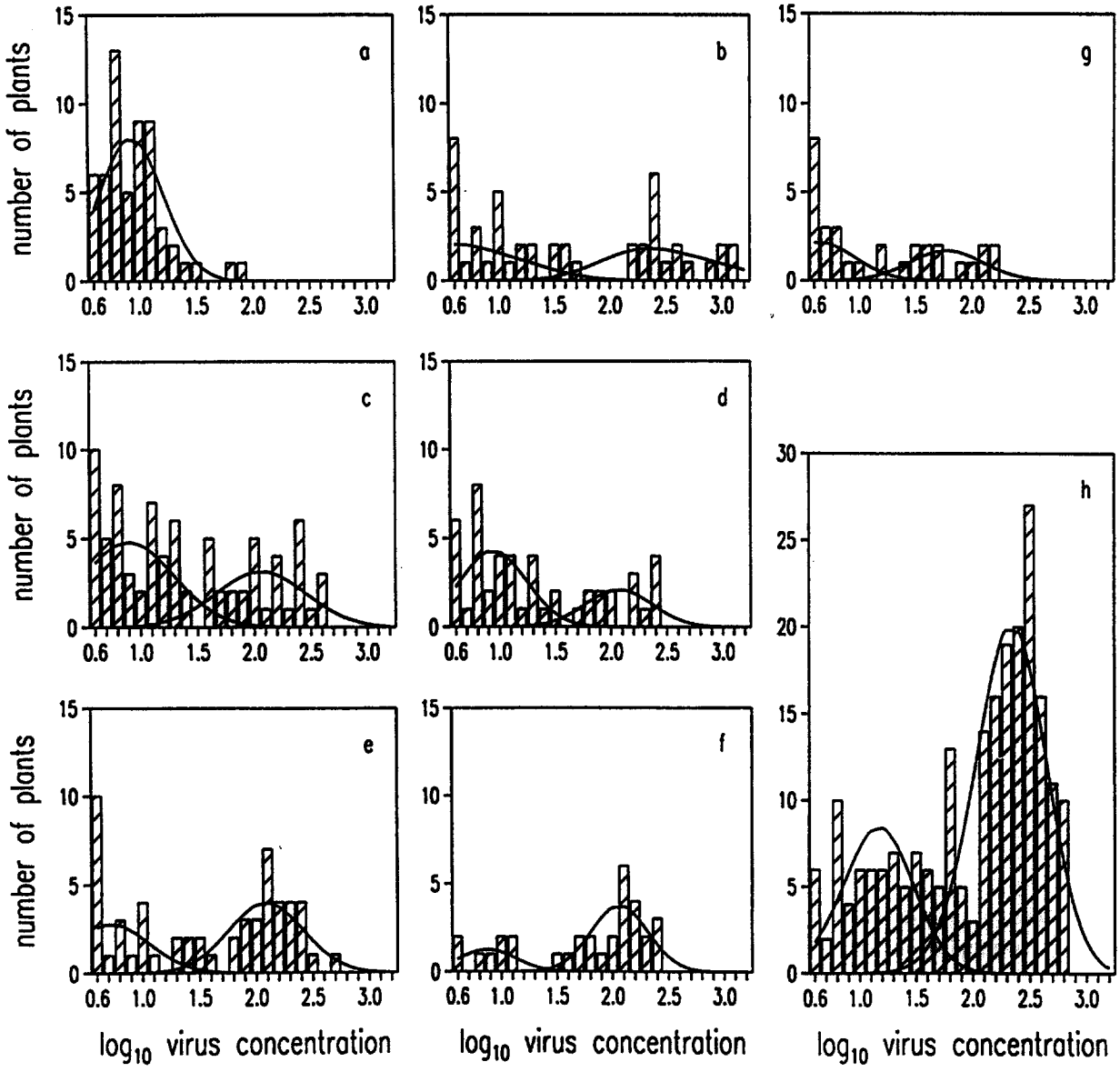

Figure 3. Curves fitted to histograms of $\log_{10}$ BNYVV concentrations of individual plants originating from crosses with WB42: F1 (91.05) (a), F1 (91.32) (b), F2 (92.04) (c), F2 (92.14) (d), F2 (93.38) (e), BC (93.38) (f), F2 (92.26) (g) and BC (92.26) (h).

by two unlinked, independent dominant major genes, or by two unlinked complementary dominant major genes, both required for resistance and also a hypothesis concerning distorted segregation of one (or more) dominant gene(s) was taken into account.

Based on similar mean virus concentrations in the resistant plants of the F1 families as in resistant plants of WB42, it was concluded that resistance in WB42 inherits dominantly. Mixture models were used to estimate the segregation ratios of resistant and susceptible plants together with the 95% confidence intervals (Table 3). The hypothesis, that resistance in WB42 is based on one dominant major gene, was accepted for the F1 and most of the F2 families. However, for three BC families this hypothesis was rejected due to the large number of susceptible plants. Also the hypothesis, that resistance was based on two unliked, independent dominant major genes, was rejected. If two genes for resistance to BNYVV were acting as complementary genes, as was proposed in the third hypothesis, the

Table 2. The observed segregation ratios of resistant (R) to susceptible (S) plants based on mixture models together with the 95% confidence intervals demonstrate the possible fit of the expected ratios in crosses with Holly-1-4, using the hypothesis that resistance to BNYVV is controlled by a single dominant major gene

Plant materials[a]	Number of plants	Mean observed ratios R : S	95% confidence intervals[b]	Expected ratio R : S	Hypothesis accepted
F1(91.11)[c]	48	1 : 0	–	1 : 0	yes
F1(91.38)	32	1 : 0	–	1 : 0	yes
F2(92.01)	80	0.74 : 0.26	± 0.08	3 : 1	yes
F2(92.03)	80	0.78 : 0.22	± 0.06	3 : 1	yes
F2(92.11)	80	0.83 : 0.17	± 0.04	3 : 1	no
F2(92.13)	32	0.69 : 0.31	± 0.20	3 : 1	yes
BC(92.03)	135	0.44 : 0.56	± 0.12	1 : 1	yes
BC(92.13)	31	0.51 : 0.49	± 0.25	1 : 1	yes

[a] Identification number of the crosses in parentheses.
[b] The 95% confidence interval is based on the mean observed ratios ± 1.96 * standard error. If 0.75 fits within the 95% confidence interval for resistance, it means that the hypothesis is accepted that 75% of the plants can be resistant.
[c] Resistant F1 plants were selected from F1(91.11) for the production of F2 and BC families.

95% confidence intervals contained all the expected segregation ratios for F2 and BC families. Therefore, the hypothesis that resistance to BNYVV in WB42 is based upon the existence of two complementary genes is accepted as a possible explanation.

Distortion of segregation of one (or more) dominant major gene(s) for resistance could be another plausible explanation for the observed segregation ratios in the segregating families of WB42. Then, almost all observed segregation ratios can be regarded as being the result of a selective advantage for *vulgaris* resulting in a surplus of susceptible plants. The observed segregation ratio for resistant to susceptible plants in the four BC families was estimated to be 0.28 (Rr) : 0.72 (rr), which could be the result of distortion in the male gametes. If only one resistance gene was involved, distortion in the F2 families would result in an expected ratio for resistant to susceptible plants as 0.64 (RR and Rr) : 0.36 (rr). This fits very well with most of the observed segregation ratios, and therefore, the hypothesis that the inheritance of resistance could be explained by one major gene that segregated distorted might be accepted for WB42.

Discussion

Resistance to viruses is often evaluated by the expression of disease symptoms, without estimating any virus multiplication (Fraser, 1990). Plants will then be classified as resistant in case no symptoms or necrotic lesions appear or as susceptible when symptoms appear. Typical symptoms of rhizomania infection, like proliferation of lateral roots, constriction of the tap root and browning of the vascular system, can only be analyzed after the growth of sugar beet plants in the fields for several months. To avoid such long testing periods and to create the possibility of analysing wild beets as well, greenhouse experiments were conducted in which the multiplication of BNYVV was measured as a parameter of resistance (Bürcky & Büttner, 1985; Giunchedi et al., 1985; Whitney, 1986; Paul et al., 1992). The Holly source and also WB42 were not immune to BNYVV multiplication, since several resistant plants contained detectable virus concentrations (Lewellen et al., 1987; Whitney, 1989).

Inheritance of resistance to BNYVV in Holly-1-4 and WB42 was studied in segregating F2 or BC families. Gene-dosage effects were not found as virus concentrations of resistant F1 plants were similar to those in resistant parents. These results demonstrated the dominant character of the resistance. Classification of plants from the segregating F1 families and the following F2 and BC generations into resistant or susceptible, however, was complicated due to the presence of plants with intermediate virus concentrations. Differences in data originating from field tests could be due to the irregular infestation of a field, resulting in differential infections at various parts of the roots (Lewellen & Biancardi, 1990). In greenhouse tests

Table 3. The observed segregation ratios of resistant (R) to susceptible plants (S) based on mixture models together with the 95% confidence interval for the resistant plants demonstrate the possible fit of the expected ratios in crosses with WB42, using the hypotheses that resistance to BNYVV is controlled by one dominant major gene (1), by two unlinked and independent dominant major genes (2) or by two unlinked complementary dominant major genes, both required for resistance (3)

Genotype WB42				One dominant major gene		Two unlinked and independent dominant major genes				Two complementary dominant major genes	
				RR or Rr		$R_1R_1R_2R_2$ or $R_1r_1R_2R_2$ or $R_1R_1R_2r_2$ or $R_1r_1R_2r_2$				$R_1R_1R_2R_2$ or $R_1R_1R_2r_2$ or $R_1r_1R_2R_2$ or $R_1r_1R_2r_2$	
Genotype resistant F1				Rr		$R_1r_1R_2r_2$		$R_1r_1r_2r_2$ or $r_1r_1R_2r_2$		$R_1r_1R_2r_2$	
Plant materials[a]	Number of plants	Observed ratio R : S	95% confidence intervals[b]	Expected ratio R : S	Hypothesis accepted	Expected ratio R : S	Hypothesis accepted	Expected ratio R : S	Hypothesis accepted	Expected ratio R : S	Hypothesis accepted
F1(91.05)[c]	57	1 : 0	–	1 : 0	yes	1 : 0	yes	1 : 0	yes	1 : 0	yes
F1(91.14)	64	0.57 : 0.43	± 0.18	1 : 1	yes	15 : 1	no	3 : 1	no	1 : 1	yes
F1(91.32)	47	0.53 : 0.47	± 0.22	1 : 1	yes	15 : 1	no	3 : 1	no	1 : 1	yes
F2(92.04)	79	0.60 : 0.40	± 0.16	3 : 1	yes	15 : 1	no	3 : 1	yes	9 : 7	yes
F2(92.06)	32	0.50 : 0.50	± 0.29	3 : 1	yes	15 : 1	no	3 : 1	yes	9 : 7	yes
F2(92.14)	48	0.67 : 0.33	± 0.14	3 : 1	yes	15 : 1	no	3 : 1	yes	9 : 7	yes
F2(92.26)	31	0.56 : 0.44	± 0.25	3 : 1	yes	15 : 1	no	3 : 1	yes	9 : 7	yes
F2(93.38)	56	0.41 : 0.59	± 0.18	3 : 1	no	15 : 1	no	3 : 1	no	9 : 7	yes
F2(93.39)	32	0.54 : 0.46	± 0.27	3 : 1	yes	15 : 1	no	3 : 1	yes	9 : 7	yes
BC(92.06)	32	0.20 : 0.80	± 0.08	1 : 1	no	3 : 1	no	1 : 1	no	1 : 3	yes
BC(92.26)	224	0.30 : 0.70	± 0.06	1 : 1	no	3 : 1	no	1 : 1	no	1 : 3	yes
BC(93.38)	32	0.25 : 0.75	± 0.12	1 : 1	no	3 : 1	no	1 : 1	no	1 : 3	yes
BC(93.39)	32	0.37 : 0.63	± 0.22	1 : 1	yes	3 : 1	no	1 : 1	yes	1 : 3	yes

[a] Identification number of the crosses in parentheses.
[b] The 95% confidence interval is based on the mean observed ratios ± 1.96 * standard error. If 0.75 fits within the 95% confidence interval for resistance, it means that the hypothesis is accepted that 75% of the plants can be resistant.
[c] Resistant F1 plants were selected from F1(91.14) for the production of F2 and BC families 92.04, 92.06, 92.14 and 92.26, and from F1(91.05) for F2 and BC families 93.38 and 93.39.

susceptible plants of the cultivars 'Regina', 'Queen' and the accession MS-2 always contained high virus concentrations, compared to the resistant accessions Holly-1-4 and WB42. However, for genetical analysis it is important to realise that any variation in the environment may interact with genotypic variation to produce phenotypic variation, which is not explicable in genetic terms alone (Fraser, 1990). Besides, also minor genes could be involved in the expression of resistance in these plants.

Studying the inheritance of resistance to potato leafroll virus in potato accessions Barker et al. (1994) demonstrated the usefulness of mixture models for the discrimination of plants with intermediate levels of virus multiplication for the classification in resistant and susceptible plants. In this study mixture models (Jansen, 1993, 1994) were used to analyze the inheritance of resistance to BNYVV. Resistance in Holly-1-4 was characterised as being monogenic, and thus confirmed earlier suggestions by Lewellen et al. (1987). Inheritance of resistance to BNYVV in *B. vulgaris* subsp. *maritima* WB42, was more complicated. However, using the procedures of the mixture models resulted in rejecting the hypotheses that resistance to BNYVV from WB42 was simply based on one or two dominant major genes. Simultaneously, linkage of two dominant resistant genes can also be rejected, as the expected ratios for resistant and susceptible plants are somewhere between the ratios found for one or two unlinked and independent genes, whereas the observed ratios showed a surplus of susceptible plants. Two other hypotheses, viz. two complementary genes or male gametic distortion of segragation of one or more major genes, are both fitting the observed results. However, inheritance to viruses based on two complementary genes has been described only rarely. Therefore, it is thought that the hypothesis which suggests distorted segregation of one or more dominant major genes for resistance located on the *maritima* chromosome seems to be more plausible. Such distortion of segregation was also described before for certain isozymes in crosses between *B. vulgaris* or *B. vulgaris* subsp. *maritima* with *B. macrocarpa* (Abe & Tsuda, 1988), whereas Aicher & Saunders (1990), Wagner & Wricke (1991), Wagner et al. (1992) and Abe et al. (1993) detected distortion of segregation of isozymes or morphological markers in crosses between different sugar beets. Reciprocal differences were observed by Abe & Tsuda (1988) when the F1, which contained chromosomes of *B. macrocarpa*, was used as a pollinator. This could possibly be explained by selective elimination of male gametes as a result of pollen sterility, certation or incompatibility. Another explanation is distortion of segregation caused by zygotic selection, due to differences in fitness between the zygotes (Wagner et al., 1992).

The present study also demonstrated differences in the levels of resistance, as the average virus concentrations in the rootlets of resistant plants of Holly-1-4 and the progenies of Holly-1-4 were higher than in WB42 and the progenies of WB42. After incubating seedlings of Holly-1-4 and WB42 in viruliferous zoospore suspensions, the difference in virus multiplication between these resistant accessions was even more clear (Paul et al., 1993c), since only WB42 could then be discriminated from susceptible controls, which contained high virus concentrations. It is likely that differences between Holly-1-4 and WB42 can be explained by the existence of different alleles or genes in these accessions, or by the presence of various minor genes in the background, resulting in different mechanisms of resistance. The relative performance of susceptible and partially resistant cultivars in infested fields has been shown to be related to the level of resistance determined in a greenhouse experiment (Paul et al., 1993a), and therefore, it can be expected that differences in virus multiplication in the greenhouse between families with resistance from Holly-1-4 or from WB42 will be expressed in the field.

Although resistance to plant viruses is often under simple genetic control, involving only a single locus, independent genes at two loci were found as well (Fraser, 1990). Combining different genes for resistance to BNYVV could be very useful for sugar beet breeders to obtain varieties with higher levels of resistance to rhizomania, since in general monogenic virus resistance can easily be broken, as a result of high mutation frequencies of the virus genome (Spaar et al., 1992). In a small number of cases genes for resistance, like the *Tm-2*^{2} gene in tomato against tomato mosaic virus, the R_y gene in potato against potato virus Y and the *N* gene against TMV in tobacco, have proven to be outstandingly durable in practice (Fraser, 1990). Therefore, both the Holly source of resistance to BNYVV and sources such as WB42 should be used in breeding programmes. Currently studies on allelism are performed, with plants originating from crosses between Holly-1-4 and WB42, together with the search for molecular markers, to determine whether different alleles or genes for resistance are involved in Holly-1-4 and WB42.

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